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Short report

Pattern and outcome of *Cleistanthus collinus* (Oduvanthalai) poisoning in a tertiary care teaching hospital in South India

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ABSTRACT

There is paucity of information on human studies about *Cleistanthus collinus* (Oduvanthalai) poisoning at global level. The present study was done to find out the pattern and outcomes with acute poisoning of this plant poison. Retrospective record based study was conducted among acute *C. collinus* (Oduvanthalai) poisoning cases admitted between January 2010 and December 2010 in a tertiary care teaching hospital in South India. A total of 51 cases were analyzed with 52.9% of them being females and 51% belonged to 21–40 years age group. Interpersonal conflict was the stressor for poisoning in 76% cases. Mortality rate was 17.6% with a median duration of 3.5 days from time of ingestion. Majority of the patients who died during hospitalization had ingested decoction (77.8%), and had neurological manifestations (77.8%), hypokalemia (77.8%), neutrophilia (66.7%), leucocytosis (55.6%) and elevated blood urea (77.8%). It was found that lower potassium level, white blood cell and neutrophil count were significantly associated with mortality due to poisoning.

1. Introduction

Cleistanthus collinus also known as *Oduvanthalai* in Tamil is the most common poisoning with plant products in southern India.[1] It is also popularly known as *Garari* in Hindi; *Vadise* in Telugu; *Nilapala* in Malayalam and *Karlajuri* in Bengali in different parts of India.[2] Though the plant is widely distributed, poisoning with *C. collinus* (Oduvanthalai) has not been reported from other parts of India or elsewhere in the world in English literature. All parts of the plant are poisonous and principal toxins extracted from various parts of the plants are lignan lactones, diphyllin and glycosides such as Cleistanthin A and B.[3] Main clinical features of poisoning with *C. collinus* are hypokalemia, acidosis, hypotension and respiratory failure.[4,5] Active principles of the plant are shown to cause distal renal tubular acidosis and type 2 respiratory failure in rats.[6] Inhibition of proton pumps in distal renal tubular cells by the toxin causes failure of acid secretion leading to distal renal tubular acidosis thus causing kaliuresis and hypokalemia.[7,9] Hypotension caused by this poison is attributed to the α-1 adrenergic receptor antagonist effect on the peripheral vascular system.[8] Poisoning with this plant poison can be fatal with reported mortality rates varying between 12.5% and 32%.[5,9] Treatment is mainly supportive along with replacement of potassium but animal studies have revealed that N-acetylcysteine may be a promising antidote.[10,11] The present study was done to find out the recent trends in clinical features and outcome of patients admitted to a tertiary care teaching hospital with *C. collinus* (Oduvanthalai) poisoning.

2. Methodology

We conducted a retrospective analysis of data of all patients admitted to Jawaharlal Institute of Postgraduate Medical Education and Research (JIPMER), Puducherry, a tertiary care teaching hospital in southern India with acute Oduvanthalai poisoning between January 2010 and December 2010. Details of all patients regarding demographics, stressors for poisoning, parts of the plant ingested, clinical features at presentation to the hospital, routine laboratory investigation at the time of hospitalization, serum potassium concentration during stay in the hospital, treatment received during hospital stay and outcomes were noted from the retrieval of medical records. Statistical analyses were carried out using both descriptive and inferential statistics. Independent student's *t*-test

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was used to compare the laboratory parameters at admission in relation to the outcome. Chi-square test was used to compare the categorical data between the groups. All statistical analyses were carried out at 5% level of significance and p value <0.05 was considered to be statistically significant. Statistical analyses were carried out using PASW Statistics -19.0 version.

3. Results

A total of 51 patients were admitted with Oduvanthalai poisoning during the study period. Forty nine (96%) of them ingested it with suicidal intent whereas the remaining two kids aged 8 and 9 years were given the plant poison by the mother before she also took the same with suicidal intention. Around half (52.9%) of the patients were females and around half (51%) of the patients belonged to the age group of 21–40 years. Age group and sex was not found to be significant in poisoning ($p = 0.156$) (Table 1). Majority of patients (72%) sought medical attention within 24 h of ingestion of the poison whereas in the remaining 28% patients there was a delay of more than 24 h in seeking medical attention. Mean age of patients was 31.7 years with range from 8 to 75 years. Mean duration of hospitalization was 5.3 days with range from 2 to 14 days. There were 9 deaths during the study period with the mortality rate of 17.6%. Mean duration from ingestion of Oduvanthalai to death was 4.2 days with a median duration of 3.5 days.

Interpersonal conflicts in the form of fight with spouse or other family members were the stressor for self poisoning in majority (76%) of patients. Other important stressors were health, finance or school related. All patients had presented with ingestion of leaves only but in various forms such as decoction (leaves boiled in water), crushed or whole leaves as such.

Twenty nine (56.8%) patients were asymptomatic at the time of presentation and 22 (43.1%) were symptomatic. The most common symptoms at presentation were abdominal pain, vomiting and giddiness. Drowsiness and confusion was seen only in minority of patients. Cardiac manifestations were seen in 5 (9.8%) patients. Hypotension (systolic blood pressure less than 90 mm Hg) and tachycardia (heart rate $>$ 90 beats/min) was seen in 1 patient and bradycardia (heart rate $<$ 60 beats/min) was seen in 4 patients.

Mean serum potassium concentration at the time of admission was 4.0 meq/l with range of 2.2–5.8 meq/l. Hypokalemia (serum potassium less than 3.5 meq/l) was seen in 17 (33.3%) patients; 9 (17.6%) had hypokalemia at the time of admission and additional 8 (15.6%) developed it in the hospital. Twenty four hours urinary potassium concentration were available in 10 patients and renal potassium wasting (24 h urine potassium more than 15 meq/day) was seen in 5 patients.

Forty two (82.3%) patients received either oral or intravenous potassium replacement; only 9 (19.6%) patients did not receive any potassium supplementation. Only 4 (7.8%) patients received N-acetylcysteine (NAC); 1 received intravenous regimen with similar dosing as for paracetamol poisoning, 2 received 600 mg twice a day for 3 days orally and 1 received intravenous followed by oral N-acetylcysteine.

Table 1
Age and sex distribution of study subjects ($n = 51$).

Age group (in years)	Sex	
	Male (%)	Female (%)
≤20	4 (16.7)	11 (40.7)
21–40	15 (62.5)	11 (40.7)
>40	5 (20.8)	5 (18.5)
Total	24	27

$X^2 = 3.718$, $p = 0.156$.

Proportion of patients who presented with giddiness and abdominal pain was more in the group who died during hospitalization than those who survived whereas proportion of patients who presented with vomiting was more in the group who survived during hospital stay. Characteristics of patients who died are described in Table 2.

Elevated white blood cell (WBC) count and neutrophilia at the time of admission and lowest potassium concentration during hospital stay were significantly different in those who died during hospital stay than those who survived ($p = <0.05$). Blood urea was found to be higher in those who died as compared to those who survived but it was not statistically significant. Comparative laboratory investigations between those who died during hospital stay and those who died are shown in Table 3.

4. Discussion

Poisoning with parts of *C. collinus* (Oduvanthalai) plant is peculiar to southern India especially the states of Tamil Nadu and Pondicherry. It is possible that word of mouth which is restricted to pockets of rural areas might be the reason why this poisoning is localized to this part of the world. The present study has brought out some important observations. One previous study from the same institute found that mortality was 32%[5] in contrast to present study in which the mortality is 17.6%. This improvement in survival may be attributed to increased potassium replacement in the present study. More than 82.3% of patients received some form of potassium replacement in the present study even though hypokalemia was seen in only 33.3% of patients. Similar findings were seen in a recent study done by Keshavan Nampoothiri et al.[9] Cardiac manifestations were less in the present study as compared to previous studies [4,5] which also might have led to decrease in mortality. Other reasons for decrease in mortality might be due to smaller quantity of poison ingested, seeking of early medical intervention, and the use of N-acetylcystiene. Another important observation is that serum potassium concentration during hospital stay was lowest in those who died as compared to those who survived although serum potassium concentration at the time of admission was not significantly different between the groups. Therefore it is very important to monitor serum potassium concentration in patients with significant poisoning as hypokalemia can develop during hospitalization subsequently in many of them.

Leukocytosis and neutrophilia at admission also were predictive of poor survival in these patients. Presence of neurological manifestations at presentation like giddiness, drowsiness and confusion were also predictive of poor outcome whereas presence of vomiting after ingestion was protective similar to previous study.[5]

The present study is one of the biggest studies of patients with ingestion of *C. collinus* (Oduvanthalai). However, the present study also has a major limitation of being a retrospective case record based study. Other important limitation of the present study is that urinary potassium loss was documented in only 5 of

Table 2
Associated factors of mortality due to Oduvanthalai poisoning.

Associated factors	No ($n = 9$)	%
Age > 40 years	5	55.6
Ingestion of decoction	7	77.8
CNS manifestations	7	77.8
Hypokalemia	7	77.8
Leukocytosis	5	55.6
Neutrophilia	6	66.7
Elevated blood urea	7	77.8
Elevated creatinine	3	33.3

Table 3
Comparison of laboratory parameters between the groups at admission.

Laboratory parameter	Group	No	Mean	SD	*p* Value
Potassium (meq/l)	Survived	41	3.6	0.7	$p > 0.05$
	Died	9	3.3	0.9	
Lowest potassium[a] (meq/l)	Survived	39	3.2	0.6	***p* < 0.05**
	Died	9	2.3	0.5	
White blood cell count (/mm^3)	Survived	34	8727	2485	***p* < 0.05**
	Died	7	15,614	4149	
Neutrophils (%)	Survived	32	72.3	10.8	***p* < 0.05**
	Died	8	87.8	8.05	
Blood urea (mg/dl)	Survived	39	28.0	18.0	$p > 0.05$
	Died	9	35.7	11.0	
Serum creatinine (mg/dl)	Survived	31	0.6	0.8	$p > 0.05$
	Died	7	0.7	0.5	

P values in bold represents the positive correlation.
[a] Lowest potassium recorded in the hospital.

the 10 patients for whom urinary potassium values were available. Vomiting and dehydration might have contributed for hypokalemia in other patients.[2] Prospective studies with early versus as required potassium replacement and subsequently use of N-acetylcystiene in the management of poisoning with *C. collinus* (Oduvanthalai) will tell us whether the mortality can further be reduced.

5. Conclusion

C. collinus (Oduvanthalai) poisoning is common among productive age group which needs close monitoring of serum potassium and other blood parameters to prevent the complications and mortality.

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Conflict of interest
None.

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